The Egyptian Journal of Hospital Medicine (October 2025) Vol. 101, Page 4841-4848

ECG Pattern of Hyperkalemia in Acute versus Chronic Kidney Disease

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ABSTRACT

Background: Hyperkalemia is a life-threatening complication of kidney disease, but its electrocardiographic (ECG) manifestations may differ between acute Kidney Disease (AKD) and chronic kidney disease (CKD), particularly in pediatric patients.

Aim of the work: to compare ECG changes in hyperkalemic children with AKD and CKD and to correlate these findings with biochemical and clinical parameters.

Patients and Methods: A cross-sectional study was conducted on 89 children with hyperkalemia, including 40 with AKD and 49 with CKD, at the Pediatric Nephrology Unit, Menoufia University. All patients underwent detailed history, anthropometric and clinical examination, laboratory investigations (serum electrolytes, urea, creatinine), and standard 12-lead ECG.

Results: Children with CKD were older than those with AKD. Serum potassium was significantly higher in AKD, whereas urea and creatinine were markedly higher in CKD. QRS prolongation occurred more often in AKD than CKD. QTc prolongation was more frequent in AKD, with abnormal T waves also significantly higher. Small or flat P-waves were strikingly more prevalent in AKD (97.5%) compared with CKD (28.6%, $p < 0.001$). Logistic regression confirmed that AKD independently predicted P-wave abnormalities, while low calcium was also associated (OR = 0.54, $p = 0.017$). The Tp–e interval was significantly shorter in AKD than in CKD.

Conclusion: Hyperkalemia in children presents with distinct ECG abnormalities that differ between AKD and CKD. Acute cases were more frequently associated with abnormal P-waves, abnormal T-waves, QRS widening, and QTc prolongation, reflecting rapid and unstable potassium shifts. In contrast, CKD patients demonstrated more chronic adaptive changes. These findings highlight the importance of careful ECG monitoring in hyperkalemic children, particularly those with AKD, to enable timely detection of life-threatening arrhythmias and guide condition-specific management strategies.

Keywords: Hyperkalemia; Electrocardiography; Acute Kidney Injury; Chronic Kidney Disease; Pediatrics.

INTRODUCTION

Acute kidney injury (AKI) is characterized by a sudden loss of excretory kidney function and may develop within days. It is part of a broader spectrum termed acute kidney diseases and disorders (AKD), which includes persistent kidney dysfunction that may lead to chronic kidney disease (CKD) [(1)].

Chronic kidney disease (CKD) is a progressive and irreversible condition that may culminate in renal failure, requiring renal replacement therapy such as dialysis or transplantation. The term CKD has replaced older terminology such as chronic renal failure and chronic renal insufficiency, reflecting renal dysfunction as a continuum rather than a discrete state [(2)].

Hyperkalemia, defined as elevated serum potassium levels, is a common and potentially life-threatening electrolyte disturbance. Despite the presence of various management guidelines, there is no universal consensus regarding precise potassium thresholds or standardized approaches for acute and chronic hyperkalemia, highlighting an unmet need in real-world practice [(3)].

The incidence of hyperkalemia has increased with the use of renin–angiotensin–aldosterone system inhibitors, beta-blockers, and mineralocorticoid antagonists, in addition to the rising prevalence of CKD, dialysis dependency, and heart failure. Furthermore, the development of new oral potassium binders has renewed clinical interest in this condition [(4)].

Clinical recognition of hyperkalemia can be difficult, as symptoms are often nonspecific, including fatigue, muscle weakness, palpitations, chest discomfort, nausea, or dyspnea. Physical findings such as hypotension, depressed reflexes, and arrhythmias are also nonspecific, and reliance on history and examination alone may delay timely diagnosis and management [(5,6)].

Electrocardiography (ECG) remains an inexpensive, non-invasive, and widely accessible diagnostic tool. Elevated serum potassium levels above 6.0 mmol/L may be associated with characteristic ECG changes such as peaked T-waves, PR prolongation, QRS widening, loss of P- waves, ST depression, and QT interval shortening [(7)]. These abnormalities tend to become more evident with severe hyperkalemia [(7)].

However, the diagnostic reliability of ECG in predicting hyperkalemia is debated. While certain changes are highly specific, their sensitivity is low, as many hyperkalemic patients do not show classical ECG findings. Thus, the utility of ECG alone in diagnosing hyperkalemia, particularly in acute kidney injury versus chronic kidney disease, remains uncertain [(8,9)].

So, our study aimed to compare ECG changes in hyperkalemic children with AKD and CKD and to

Received: 02/05/2025
Accepted: 04/07/2025

correlate these findings with biochemical and clinical parameters.

PATIENTS AND METHODS

Study design and setting:

This cross-sectional study was conducted at the Nephrology Unit, Pediatric Department, Faculty of Medicine, Menoufia University. From April 2023 to April 2024.

Ethical considerations

The study protocol was approved by the Institutional Ethics Committee of the Faculty of Medicine, Menoufia University, IRB Number: 2/2023PEDI 18 and a written informed consent was obtained from the parents or legal guardians of all participants prior to inclusion.

Sample size estimation

Based on review of past literature [10] who found that a QRS duration of 120 ms or greater is most predictive of hyperkalemia in the End-Stage Renal Disease population (Correlation Coefficient= .35). The sample size is calculated using statistics and sample size pro program version 6 the sample size is 70. The power of study is 80% and confidence level is 95%.

Study population

Children with kidney diseases were enrolled and categorized into two groups:

- **Group I (CKD group):** 49 Children diagnosed with chronic kidney disease according to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 criteria, defined as the presence of kidney damage markers or reduced glomerular filtration rate (GFR <60 mL/min/1.73 m²) persisting for more than three months.
- **Group II (AKI/AKD group):** 40 Children diagnosed with acute kidney injury or acute kidney disease/disorders (AKD) based on KDIGO 2021 guidelines, which define AKI as a rise in serum creatinine ≥0.3 mg/dL within 48 hours, or ≥50% within 7 days, or urine output <0.5 mL/kg/h for 6 hours, and AKD as kidney dysfunction lasting <3 months.

Data collection

All participants underwent the following assessments:

- **History and anthropometry:** Age, sex, weight, height, and disease history (onset, duration, course). Weight and height were measured using standardized methods and interpreted according to Egyptian Z-score reference charts.

  Clinical examination: General (vital signs) and systemic (abdominal, cardiac, chest, and neurological) examination were performed.

  - **Laboratory investigations:** Complete blood count (CBC), serum electrolytes (sodium, potassium, calcium, phosphorus), and renal function tests (urea, creatinine) were measured using standard automated analyzers.

Electrocardiography (ECG): A standard 12-lead ECG was performed for each patient using AKAI ECG machine (AKAI ECG machine(CG3T)_AKAI-MED Hong Kong)with athree-channel electrocardiographic recorder at a paper speed of 25 mm/s and calibration of 10 mm/mV. ECG parameters assessed included rhythm, heart rate, QRS axis, P-wave duration, PR interval, QT and corrected QT (QTc) intervals, and T-peak to T-end interval. The QTc was calculated using Bazett's formula. Normal values were interpreted according to age-adjusted pediatric reference ranges.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics for Windows, version 20.0 (Armonk, NY: IBM Corp., released 2011). Qualitative variables were presented as frequencies and percentages, and quantitative variables were expressed as mean ± standard deviation (SD) for normally distributed data or median and interquartile range (IQR) for skewed data, with normality tested using the Shapiro–Wilk test. Comparisons between the AKD and CKD groups were performed using the chi-square test (with Monte Carlo correction when >20% of expected cell counts were <5), Student's *t*-test for normally distributed quantitative variables, and the Mann–Whitney U test for non-normally distributed variables. Multivariate linear regression was applied to determine independent predictors of PR interval and QTc, while binary logistic regression was used to identify factors associated with abnormal ECG parameters.

RESULTS

In the present study, 49 children with CKD and 40 with AKD were included. Males represented 61.2% of the CKD group and 70% of the AKD group, with no significant sex difference (p=0.387). Children with CKD were significantly older (mean age 11.1 ± 3.7 years) compared to those with AKD (9.3 ± 2.8 years, p=0.008). Consanguinity was present in 12.2% of CKD and 15% of AKD cases (p=0.705). Serum sodium was higher in CKD (138.6 ± 2.7 mEq/L) than AKD (135.0 ± 10.8 mEq/L, p=0.042), while potassium was significantly lower in CKD (5.95 ± 0.23 vs. 6.15 ± 0.38 mEq/L, p=0.006). Urea (86.4 ± 39.4 vs. 52.3 ± 33.2 mg/dL, p<0.001) and creatinine (6.7 ± 3.1 vs. 1.4 ± 0.3 mg/dL, p<0.001) were markedly higher in CKD compared to AKD. Growth assessment revealed that 52.1% of CKD patients were stunted versus none in AKD (p<0.001), and BMI was also significantly higher in CKD (27.3 ± 4.8 vs. 24.1 ± 3.3, p<0.001). **(Table: 1).**

Table (1): Comparison between the two studied groups according to demographic and Lab data

	CKD (n = 49)		AKD (n = 40)		Test of Sig.	P-value
	No.	%	No.	%		
Sex						
Male	30	61.2	28	70.0	χ^2= 0.747	0.387
Female	19	38.8	12	30.0		
Age (years)						
Min. – Max.	4.0 – 17.0		4.0 – 14.0		t= 2.719*	0.008*
Mean ± SD.	11.12 ± 3.73		9.25 ± 2.76			
Median (IQR)	12.0 (8.0 – 14.0)		10.0 (7.50 – 11.0)			
Consangunity						
Negative	43	87.8	34	85.0	χ^2= 0.143	0.705
Positive	6	12.2	6	15.0		
Weight	**(n = 49)**		**(n = 40)**			
Min. – Max.	20.0 – 75.0		23.0 – 60.0		t=1.598	0.114
Mean ± SD.	43.51 ± 13.99		39.50 ± 9.61			
Median (IQR)	45.0 (30.0 – 50.0)		40.0 (30.50 – 47.50)			
Weight Z score	**(n = 49)**		**(n = 40)**			
Normal	32	65.3	27	67.5	□²= □□□□□	0.652
Under weight	8	16.3	4	10.0		
Over Weight	9	18.4	9	22.5		
Height	**(n = 48#)**		**(n = 40)**			
Min. – Max.	90.0 – 162.0		100.0 – 150.0		t=0.849	0.398
Mean ± SD.	124.4 ± 18.08		127.4 ± 14.07			
Median (IQR)	125.0 (110.0 – 139.50)		130.0 (118.0 – 139.50)			
Height Z score	**(n = 48)**		**(n = 40)**			
Normal	23	47.9	40	100.0	□²= □□□□□□□	<0.001*
Stunt	25	52.1	0	0.0		
Body mass index	**(n = 48)**		**(n = 40)**			
Min. – Max.	19.20 – 37.0		17.90 – 31.30		t=3.672*	<0.001*
Mean ± SD.	27.28 ± 4.80		24.08 ± 3.34			
Median (IQR)	26.75 (23.09 – 31.0)		23.60 (21.65 – 26.45)			
Body mass index Z score	**(n = 48)**		**(n = 40)**			
Normal	26	54.2	27	67.5	X^2= 5.733	^{MC}p= 0.129
Under weight	3	6.3	3	7.5		
Over Weight	8	16.7	8	20.0		
Obesity	11	22.9	2	5.0		
Na (mmol/L)						
Mean ± SD.	138.6 ± 2.71		135.0 ± 10.79		t= 2.091*	0.042*
K (mmol/L)						
Mean ± SD.	5.95 ± 0.23		6.15 ± 0.38		t= 2.851*	0.006*
Ca total mg/dL						
Mean ± SD.	8.43 ± 1.52		8.60 ± 0.69		t= 0.661	0.511
Urea mg/dL						
Mean ± SD.	86.41 ± 9.43		52.28 ± 3.24		U= 499.500	<0.001*
Creatinine mg/dL						
Mean ± SD.	6.70 ± 1.09		1.36 ± 0.28		t=12.044*	<0.001*

Analysis of ECG parameters revealed notable differences between the two groups. Prolonged QRS complexes were more frequent in AKD patients (42.5%) compared with CKD patients (24.5%), while normal QRS was observed in 75.5% of CKD versus 55.0% of AKD children (p=0.053). Prolonged PR intervals occurred in 47.5% of AKD and 40.8% of CKD, with no significant difference (p=0.527). QTc prolongation was detected in 27.5% of AKD compared with 12.2% of CKD patients, with mean QTc significantly longer in AKD (0.44 ± 0.06 vs. 0.41 ± 0.04 sec, p=0.035). Abnormal T-waves were more prevalent in AKD (65.0%) compared with CKD (40.8%, p=0.023). P-wave abnormalities were strikingly higher in AKD, where 97.5% had small or flat P- waves versus only 28.6% in CKD (p<0.001). Furthermore, the Tp-e interval was significantly shorter in AKD (0.06 ± 0.02 sec) compared to CKD (0.08 ± 0.02 sec, p=0.002), suggesting differences in re-polarization dynamics between groups. **(Table 2).**

Table (2): Comparison between the two studied groups according to different ECG parameters

	CKD (n = 49)		AKD (n = 40)		Test of Sig.	P-value
	No.	%	No.	%		
QRS						
Normal	37	75.5	22	55.0	χ^2= 4.701	^{MC}p= 0.053
Short	0	0.0	1	2.5		
Prolonged	12	24.5	17	42.5		
QRS duration second						
Min. – Max.	0.06 – 0.70		0.06 – 0.12		U= 842.000	0.208
Mean ± SD.	0.10 ± 0.09		0.08 ± 0.02			
Median (IQR)	0.08 (0.08 – 0.08)		0.08 (0.06 – 0.09)			
PR interval						
Not prolonged	29	59.2	21	52.5	χ^2= 0.400	0.527
Prolonged	20	40.8	19	47.5		
P.R interval second	**(n = 47)**		**(n = 40)**			
Min. – Max.	0.10 – 0.80		0.06 – 0.24		U= 823.500	0.317
Mean ± SD.	0.18 ± 0.11		0.16 ± 0.03			
Median (IQR)	0.14 (0.12 – 0.20)		0.16 (0.15 – 0.18)			
QTC						
Not prolonged	43	87.8	29	72.5	χ^2= 3.317	0.069
Prolonged	6	12.2	11	27.5		
QTC Second						
Min. – Max.	0.32 – 0.55		0.37 – 0.57		t= 2.162	0.035*
Mean ± SD.	0.41 ± 0.04		0.44 ± 0.06			
Median (IQR)	0.42 (0.39 – 0.43)		0.42 (0.39 – 0.48)			
T-wave						
Normal	29	59.2	14	35.0	χ^2= 5.158*	0.023*
Abnormal	20	40.8	26	65.0		
P-wave						
Normal	35	71.4	1	2.5	43.438*	<0.001*
Small to flat	14	28.6	39	97.5		
Tp.e second						
Min. – Max.	0.04 – 0.10		0.04 – 0.09		3.238*	0.002*
Mean ± SD.	0.08 ± 0.02		0.06 ± 0.02			
Median (IQR)	0.08(0.07 – 0.09)		0.06(0.04 – 0.08)			

Multivariate linear regression analysis for ECG parameters is presented in **(Table: 3)**. For PR interval, none of the studied biochemical variables, including serum potassium, calcium, urea, or creatinine, showed a significant independent effect, and the overall model was not statistically significant ($p = 0.810$). In contrast, for QTc interval, being in the AKD group was found to be a significant positive predictor ($\beta = 0.336$, $p = 0.049$), while serum potassium, calcium, urea, and creatinine did not show independent associations. This indicates that QTc prolongation was more strongly related to the disease category (AKD vs. CKD) rather than to individual biochemical parameters.

Table (3): Multivariate Linear regression for PR interval and QTC

for PR interval					
	B	**SE**	**Beta**	**t**	**P-value**
(Constant)	0.101	0.197		0.515	0.608
K	0.022	0.030	0.085	0.733	0.466
Ca total	0.002	0.008	0.023	0.196	0.845
Urea	0.000	0.000	-0.078	-0.574	0.568
Creatinine	-0.003	0.004	-0.106	-0.577	0.566
AKD vs CKD	-0.038	0.030	-0.231	-1.286	0.202
R^2 = 0.027 , adjusted R^2= -0.033, SE = 0.085 , F = 0.452 , p =0.810					
for QTC					
(Constant)	0.424	0.113		3.771	<0.001*
K	-0.012	0.018	-0.077	-0.689	0.493
Ca total	0.003	0.004	0.083	0.748	0.457
Urea	0.000	0.000	-0.163	-1.254	0.213
Creat	0.003	0.002	0.189	1.081	0.283
AKD vs CKD	0.033	0.017	0.336	1.986	0.049*
R^2 = 0.099 , adjusted R^2= 0.043, SE = 0.048 , F = 1.761 , p =0.130					

Multivariate logistic regression identified serum potassium as an independent predictor of PR interval prolongation, with hyperkalemia increasing the odds more than seven-fold (OR = 7.62, 95% CI: 1.44–40.33, p = 0.017). No significant predictors were observed for QTc prolongation, although the AKD group showed a non significant trend toward higher odds (OR = 4.85, p = 0.126). Abnormal T-wave morphology was significantly associated with elevated serum urea, with each unit increase raising the odds by 2.3% (OR = 1.023, 95% CI: 1.006–1.040, p = 0.006). For P-wave abnormalities, low serum calcium was an independent predictor (OR = 0.54, 95% CI: 0.32–0.89, p = 0.017), while being in the AKD group increased the odds of small or flat P-waves more than sixty-fold compared to CKD (OR = 60.43, 95% CI: 4.44–821.86, p = 0.002). These findings underscore potassium, urea, calcium, and disease category as important determinants of specific ECG abnormalities in hyperkalemic children **(Table: 4).**

Table (4): Multivariate analysis binary logistic regression for ECG parameters (Normal vs Abnormal)

for PR interval						
	B	**SE**	**Sig.**	**OR**	**95% CI**	
					LL	**UL**
K	2.031	0.850	0.017*	7.621	1.440	40.331
Ca total	-0.096	0.197	0.628	0.909	0.618	1.337
Urea	-0.002	0.007	0.750	0.998	0.984	1.012
Creat	0.151	0.113	0.181	1.163	0.932	1.452
AKD vs CKD	0.664	0.751	0.376	1.943	0.446	8.462
for QTC						
K	-0.124	0.907	0.891	0.883	0.149	5.226
Ca total	0.186	0.326	0.569	1.204	0.636	2.279
Urea	-0.011	0.009	0.236	0.989	0.971	1.007
Creatinine	0.164	0.140	0.240	1.178	0.896	1.549
AKD vs CKD	1.578	1.031	0.126	4.847	0.642	36.592
for T-wave						
K	0.340	0.835	0.684	1.405	0.273	7.222
Ca total	-0.183	0.205	0.371	0.833	0.558	1.244
Urea	0.023	0.008	0.006*	1.023	1.006	1.040
Creatinine	-0.156	0.122	0.202	0.856	0.673	1.087
AKD vs CKD	1.067	0.758	0.159	2.906	0.658	12.837
at p-wave vs normal p-wave						
K	-0.624	1.368	0.648	0.536	0.037	7.824
Ca total	-0.626	0.261	0.017*	0.535	0.321	0.892
Urea	0.004	0.011	0.722	1.004	0.983	1.026
Creat	-0.206	0.158	0.191	0.814	0.597	1.109
AKD vs CKD	4.101	1.332	0.002*	60.430	4.443	821.856

DISCUSSION

Electrocardiographic (ECG) changes associated with hyperkalemia are well-documented in the general population; however, their manifestation in pediatric patients with kidney disease particularly when comparing acute (AKD) to chronic kidney disease (CKD) remains less clearly defined [11]. Children with impaired renal function are especially vulnerable to electrolyte disturbances, with hyperkalemia being a potentially life-threatening complication. The ECG serves as a rapid, non-invasive tool for assessing cardiac involvement, yet its sensitivity and specificity in relation to serum potassium levels in different renal conditions is still debated [12]. This study aimed to evaluate and compare the ECG patterns of hyperkalemia in children with AKD and CKD and to correlate these findings with biochemical and clinical parameters to improve early detection and risk stratification.

Regarding QRS duration, our study showed a higher frequency of QRS prolongation in AKD patients (42.5%) compared to CKD patients (24.5%), approaching statistical significance. This is supported by the findings of **Simon *et al*.** who reported that ECG changes such as QRS widening tend to appear earlier and more severely in acute hyperkalemia due to the abrupt rise in extracellular potassium, leading to slowed ventricular conduction [13]. In contrast, patients with CKD may experience a more gradual rise in potassium, allowing for partial myocardial accommodation, thus explaining the lower incidence of QRS prolongation in this group. However, **Daly *et al*.** also noted that while QRS widening is a recognized manifestation of hyperkalemia, its presence is not universal and may be absent in some cases even with severe hyperkalemia, reflecting individual variability [14].

In our comparison of PR interval prolongation, no significant difference was found between AKD and CKD patients. This finding is consistent with previous research, including that of **Suarez-Rivera *et al*.**, which highlighted that PR interval changes in hyperkalemia are relatively non-specific and may occur across different stages of kidney disease [15]. Their findings emphasized that although PR prolongation can occur due to slowed atrioventricular conduction, it lacks diagnostic reliability, especially in children or patients with underlying cardiac or renal conditions.

One of the significant findings in our study was the statistically prolonged corrected QT interval (QTc) in AKD patients compared to CKD patients. While QTc prolongation is not typically associated with hyperkalemia, our finding is not unprecedented. **Golzari *et al*.** have reported that in certain clinical settings, such as rapid shifts in serum potassium levels or the presence of coexisting electrolyte disturbances (e.g., hypocalcemia or hypomagnesemia), QTc prolongation can occur [16]. In our study, the higher prevalence of prolonged QTc in AKD patients may reflect the acute and unstable biochemical environment, as well as potential multi-factorial effects on cardiac re-polarization. This contradicts the classical expectation of shortened QTc in hyperkalemia but aligns with newer evidence indicating variability in ECG response depending on the rapidity of potassium rise and other metabolic factors.

In our study, we performed multivariate regression analyses to explore the independent predictors of ECG abnormalities—specifically PR interval and QTc prolongation—in pediatric patients with hyperkalemia due to acute or chronic kidney disease. The linear regression model for the PR interval revealed that none of the variables, including serum potassium, calcium, urea, creatinine, or disease type (AKD vs. CKD), significantly predicted PR interval duration. The low R^2 value (0.027) indicates that these biochemical and clinical variables accounted for very little of the variability in PR interval. This finding is consistent with previous reports by **Barbance *et al*.** who emphasized that PR interval prolongation in hyperkalemia is not consistently correlated with serum potassium levels, particularly in pediatric or chronically ill populations where multiple factors may affect atrioventricular conduction [17].

However, logistic regression analysis for PR interval (prolonged vs. non-prolonged) revealed a statistically significant association between serum potassium and PR interval prolongation (OR = 7.62, p = 0.017), suggesting that patients with higher potassium levels are significantly more likely to exhibit prolonged PR intervals. This supports earlier work by **Tafreshi *et al*.** who identified PR interval prolongation as a potentially early ECG manifestation of hyperkalemia, although not uniformly present [18]. Our finding highlights that while serum potassium may not directly predict the degree of PR prolongation on a continuous scale, it significantly increases the likelihood of it occurring — particularly when hyperkalemia reaches clinically significant thresholds.

Regarding the QTc interval, linear regression analysis showed that the type of kidney disease (AKD vs. CKD) was a statistically significant predictor of QTc prolongation, with AKD patients more likely to have prolonged QTc. This aligns with our earlier findings and supports the hypothesis that acute, rapidly rising potassium levels in AKD may exert a more disruptive effect on ventricular re-polarization than the gradual hyperkalemia seen in CKD [19,20]. While traditional teaching suggests that hyperkalemia is more likely to shorten the QT interval, several recent studies, including those by **Tafreshi *et al*.** and others, have demonstrated that QTc prolongation can still occur in hyperkalemic patients, especially when compounded by other metabolic abnormalities such as uremia or hypocalcemia [18]. In contrast, serum potassium itself was not a significant independent predictor of QTc duration in our model, nor was it significantly associated with QTc prolongation in the logistic regression analysis. This supports the argument that

QTc prolongation in hyperkalemic patients may be multifactorial and not solely dependent on potassium concentration.

Our study revealed several significant differences in laboratory findings between children with hyperkalemia in the context of acute kidney disease (AKD) versus chronic kidney disease (CKD), shedding light on the distinct biochemical profiles associated with each condition. Serum potassium levels were significantly higher in AKD patients compared to CKD patients, likely reflecting the abrupt loss of renal excretory function in AKD, which leads to rapid accumulation of potassium. This finding aligns with **Piner *et al*.** and **Shroff *et al*.** who emphasized that acute elevations in serum potassium are more likely to result in severe and sudden ECG manifestations due to the lack of compensatory adaptation, unlike in CKD where chronic exposure allows some myocardial accommodation [21, 22].

Sodium levels were also significantly lower in AKD patients, possibly due to fluid overload, dilutional hyponatremia, or impaired sodium re-absorption in the setting of acute tubular injury. This is consistent with the findings of **Kopač. ,** who noted that AKI (acute kidney injury) in children is often accompanied by hyponatremia, especially when associated with systemic inflammatory states [23]. In contrast, CKD patients had relatively stable sodium levels, reflecting their better chronic homeostatic control.

Urea and creatinine levels were significantly higher in CKD compared to AKD (both $p < 0.001$), which is expected due to the progressive and irreversible decline in kidney function over time in CKD patients. This finding is in accordance with traditional renal physiology and corroborated by studies such as those by **Bennett *et al*.** who reported that blood urea and creatinine levels serve as distinguishing markers between acute and chronic kidney dysfunction in pediatric patients [24]. Interestingly, although AKD patients had lower absolute creatinine levels, their serum potassium was higher, again highlighting the disproportionate severity of electrolyte derangement in acute renal failure.

Our study demonstrated significant differences in ECG abnormalities between hyperkalemic children with AKD and those with CKD. One of the most striking findings was the predominance of small or flat P-waves among AKD patients (97.5%) compared to only 28.6% in CKD. Multivariate analysis confirmed that AKD was an independent predictor of P-wave abnormalities, with more than sixty-fold increased odds, while lower calcium levels were also significantly associated. These findings are in line with **Littmann and Gibbs**, who reported that P-wave attenuation and eventual disappearance are early electrophysiological consequences of severe hyperkalemia due to impaired atrial conduction [25].

QTc prolongation was another key abnormality, seen in 27.5% of AKD versus 12.2% of CKD patients, with regression analysis showing AKD itself as a significant predictor. This aligns with **Raffee *et al*.** who noted that QTc abnormalities become more prominent with severe or rapidly developing hyperkalemia, particularly in unstable patients [26].

Abnormal T-wave morphology was significantly more frequent in AKD (65.0% vs. 40.8%), and regression analysis identified serum urea as an independent predictor. While the link between uremia and T-wave abnormalities has been less frequently described, our findings suggest that azotemia may exacerbate re-polarization disturbances, in agreement with **Gilligan *et al*.**, who highlighted the complex interplay between electrolyte disturbances and uremic toxins in shaping ECG patterns [27].

Our study revealed that the Tp–e interval, a marker of ventricular re-polarization dispersion and arrhythmic risk, was significantly shorter in AKD compared to CKD. This contrasts with **Baytugan *et al*.** that identified Tp–e prolongation as an arrhythmic marker [28]. A possible explanation is that in our hyperkalemic cohort, AKD patients experienced rapid shifts in extracellular potassium, leading to accelerated re-polarization, whereas CKD patients demonstrated chronic adaptive changes that may prolong dispersion of re-polarization.

A key strength of this study is its focus on a pediatric population, where data on hyperkalemia remain limited, especially when comparing AKD and CKD. The use of comprehensive clinical, biochemical, and electrocardiographic assessments, supported by multivariate analyses, provided a multidimensional view of hyperkalemia manifestations. Nonetheless, the relatively small sample size, cross-sectional design, and lack of data on medication history may have limited the generalizability and causal interpretation of findings. Despite these limitations, the study offers valuable insights for pediatric nephrology and cardiac risk assessment.

CONCLUSION

Hyperkalemia in children presents with distinct ECG abnormalities that differ between AKD and CKD. Acute cases were more frequently associated with abnormal P-waves, abnormal T-waves, QRS widening, and QTc prolongation, reflecting rapid and unstable potassium shifts. In contrast, CKD patients demonstrated more chronic adaptive changes. These findings highlight the importance of careful ECG monitoring in hyperkalemic children, particularly those with AKD, to enable timely detection of life-threatening arrhythmia and guide condition-specific management strategies.

DECLARATIONS

Consent for publication: I certify that each author has granted permission for the work to be submitted.
Funding: No fund.
Availability of data and material: Available
Conflicts of interest: None.

Competing interests: None.

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